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Search: L7

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DATE: Friday, March 15, 2002 Printable Copy Create Case

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L6	L5	0	L6
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END OF SEARCH HISTORY

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L7: Entry 3 of 4 File: USPT Sep 4, 2001

US-PAT-NO: 6284944
DOCUMENT-IDENTIFIER: US 6284944 B1

TITLE: Gene-targeted non-human mammal with a human fad presenilin mutation and generational offspring

DATE-ISSUED: September 4, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Scott; Richard W.	West Chester	PA		
Reaume; Andrew G.	West Chester	PA		
Dorfman; Karen	West Chester	PA		

US-CL-CURRENT: 800/3; 435/455, 800/12, 800/18

CLAIMS:

What is claimed is:

1. A gene-targeted mouse heterozygous for human presenilin-1 (PS-1) mutation, said mouse comprising, in its genome, a DNA sequence encoding a functionally active PS-1 protein comprising the human P264L mutation, wherein said PS-1 protein is expressed, and wherein the A.beta.42 protein level is elevated relative to the A.beta.42 protein level in a wild-type mouse.

2. The mouse of claim 1, wherein said mutation protein encoding sequence encodes a guanosine residue at codon 265 of the mouse PS-1 encoding sequence.

3. A generational offspring of the mouse of claim 1, wherein said offspring comprises, in its genome, a DNA sequence encoding a functionally active PS-1 protein comprising the human P264L mutation, wherein said PS-1 protein is expressed, and wherein the A.beta.42 protein level is elevated relative to the A.beta.42 protein a wild-type mouse.

4. A gene-targeted mouse homozygous for human presenilin-1 (PS-1) mutation, said mouse comprising, in its genome, a DNA sequence encoding a functionally active PS-1 protein comprising the human P264L mutation, wherein said PS-1 protein is expressed, and wherein the A.beta.42 protein level is elevated relative to the A.beta.42 protein level in a wild-type mouse.

5. The mouse of claim 4, wherein said mutation protein encoding sequence encodes a guanosine residue at codon 265 of the mouse PS-1 encoding sequence.

6. A generational offspring of the mouse of claim 4, wherein said offspring comprises, in its genome, a DNA sequence encoding a functionally active PS-1 protein comprising the human P264L mutation, wherein said PS-1 protein is expressed, and wherein the A.beta.42 protein level is elevated relative to the A.beta.42 protein a wild-type mouse.

7. A method for screening a chemical compound for the ability to decrease in vivo levels of the A.beta.42 peptide, said method comprising the steps of:

(a) administering said chemical compound to the mouse of claim 6;

(b) obtaining a tissue sample from said mouse; and

(c) measuring the amount of A.beta.42 in said issue sample, wherein a decrease in the amount of A.beta.42 peptide in said issue sample compared to the amount of A.beta.42 peptide in a mouse to which said chemical compound was not administered is indicative of a chemical compound that has the ability to decrease in vivo levels of said A.beta.42 peptide.

8. The method of claim 7 wherein said tissue sample is selected from the group consisting of: brain tissue, non-brain tissue and body fluids.

9. A method for screening a chemical compound for the ability to decrease in vivo levels of the A.beta.42 peptide, said method comprising the steps of:

(a) administering said chemical compound to the mouse of claim 4;

(b) obtaining a tissue sample from said mouse; and

(c) measuring the amount of A.beta.42 in said tissue sample, wherein a decrease in the amount of A.beta.42 peptide in said tissue sample compared to the amount of A.beta.42 peptide in a mouse to which said chemical compound was not administered is indicative of a chemical compound that has the ability to decrease in vivo levels of said A.beta.42 peptide.

10. The method of claim 9 wherein said tissue sample is selected from the group consisting of: brain tissue, non-brain tissue and body fluids.

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L3: Entry 7 of 18 File: USPT Jun 12, 2001

US-PAT-NO: 6245964
DOCUMENT-IDENTIFIER: US 6245964 B1

TITLE: Transgenic rodent comprising APP-Swedish

DATE-ISSUED: June 12, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
McLonlogue; Lisa C.	San Francisco	CA		
Zhao; Jun	La Jolla	CA		
Sinha; Sukanto	San Francisco	CA		

US-CL-CURRENT: 800/12; 800/14, 800/18, 800/22, 800/3

CLAIMS:

What is claimed is:

1. A transgenic rodent that is homozygous for a diploid genome comprising a transgene integrated into said genome encoding a human APP polypeptide comprising the Swedish mutation wherein the amino acid residues at positions corresponding to positions 595 and 596 in human APP.sup.695 are asparagine and leucine, respectively, wherein the transgene is expressed to produce a human APP polypeptide having the Swedish mutation, and wherein said polypeptide is processed to ATF-betaAPP in a sufficient amount to be detectable in a brain homogenate of said transgenic rodent.

2. A transgenic rodent of claim 1, wherein the rodent is a moused.

3. A transgenic rodent of claim 1, wherein the transgene is nonhomologously integrated.

4. A transgenic rodent of claim 1, wherein the heterologous APP polypeptide comprising the Swedish mutation is expressed under the transcriptional control of a neural-specific enolase promoter.

5. A line of transgenic rodents comprising a diploid genome encoding a human APP polypeptide having the Swedish mutation wherein the amino acid residues at positions corresponding to positions 595 and 596 in human APP695 are asparagine and leucine, respectively, wherein the transgene is expressed to produce a human APP polypeptide having the Swedish mutation, and wherein said polypeptide is processed to ATF-betaAPP in a sufficient amount to be detectable in a brain homogenate of said transgenic rodent.

6. The line of claim 5, comprising a plurality of copies of the transgene.

7. The line of claim 5, produced by breeding a transgenic rodent with a wildtype rodent.

8. The line of claim 5 produced by breeding two transgenic rodents.

9. A method of producing a transgenic rodent, comprising

breeding a wildtype rodent with a transgenic rodent comprising a diploid genome encoding a human APP polypeptide having the Swedish mutation wherein the amino acid residues at positions corresponding to positions 595 and 596 in human APP695 are asparagine and leucine, respectively, wherein the transgene is expressed to produce a human APP polypeptide having the Swedish mutation, and wherein said polypeptide is processed to ATF-betaAPP in a sufficient amount to be detectable in a brain homogenate of said transgenic rodent, to produce an offspring transgenic rodent.

10. A method of producing a transgenic rodent, comprising

breeding two transgenic rodents, each comprising a diploid genome encoding a human APP polypeptide having the Swedish mutation wherein the amino acid residues at positions corresponding to positions 595 and 596 in human APP695 are asparagine and leucine, respectively, wherein the transgene is expressed to produce a human APP polypeptide having the Swedish mutation, and wherein said polypeptide is processed to ATF-betaAPP in a sufficient amount to be detectable in a brain homogenate of said transgenic rodent, to produce an offspring transgenic rodent.

11. A method of screening an agent for capacity to affect processing of amyloid precursor protein to .beta.-amyloid peptide comprising:

providing a transgenic rodent comprising a diploid genome comprising a transgene encoding a human APP polypeptide having the Swedish mutation wherein the amino acid residues at positions corresponding to position 595 and 596 in human APP695 are asparagine and leucine, respectively, wherein the transgene is expressed to produce a human APP polypeptide having the Swedish mutation, and wherein the polypeptide is processed to ATF-betaAPP in a sufficient amount to be detectable in a brain homogenate of the transgenic rodent;

contacting the transgenic rodent with the agent at a dosage of from 1 ng/kg to 10 mg/kg; and

monitoring the cleavage of the amyloid precursor protein polypeptide between the N-terminus of the .beta.-amyloid peptide and the ATF-betaAPP in the contacted transgenic rodent compared to the cleavage in a control transgenic rodent to indicate the agent affects the cleavage.

12. The method of claim 11, wherein the agent inhibits a beta-secretase activity associated with the cleavage.

13. The method of claim 11, wherein the agent causes at least a partial block of the cleavage.

14. The method of claim 11, wherein the dosage of the agent is from 10 .mu.g/kg to 1 mg/kg.

15. The method of claim 11, wherein the capacity to affect processing is a capacity to inhibit processing.

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L3: Entry 9 of 18 File: USPT Jan 16, 2001

US-PAT-NO: 6175057
DOCUMENT-IDENTIFIER: US 6175057 B1

TITLE: Transgenic mouse model of alzheimer's disease and cerebral amyloid angiopathy

DATE-ISSUED: January 16, 2001

US-CL-CURRENT: 800/12; 424/9.2, 800/18, 800/3

APPL-NO: 8/ 947295 [PALM]
DATE FILED: October 8, 1997

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L3: Entry 9 of 18 File: USPT Jan 16, 2001

US-PAT-NO: 6175057
DOCUMENT-IDENTIFIER: US 6175057 B1

TITLE: Transgenic mouse model of alzheimer's disease and cerebral amyloid angiopathy

DATE-ISSUED: January 16, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Mucke; Lennart	Foster City	CA		
Wyss-Coray; Tony	Berkeley	CA		
Masliah; Eliezer	Chula Vista	CA		

US-CL-CURRENT: 800/12; 424/9.2, 800/18, 800/3

CLAIMS:

What is claimed is:

1. A transgenic mouse comprising stably integrated into the genome of said mouse a first transgenic nucleotide sequence encoding bioactive transforming growth factor-.beta.1 (TGF-.beta.1) operably linked to a promoter and a second transgenic nucleotide sequence encoding a human amyloid precursor protein (hAPP) operably linked to a promoter, wherein said first and said second transgenic nucleotide sequences are expressed, and wherein, as a result of said expression, said transgenic mouse develops, within about three months of age, cerebrovascular amyloid deposits associated with a disease selected from the group consisting of Alzheimer's disease (AD) and cerebral amyloid angiopathy (CAA).

2. The mouse of claim 1, wherein the mouse is heterozygous for a human amyloid precursor protein (hAPP) gene product.

3. The mouse of claim 1, wherein the mouse is homozygous for a human amyloid precursor protein (hAPP) gene product.

4. The mouse of claim 1, wherein the transgenic nucleotide sequence encoding bioactive TGF-.beta.1 is overexpressed, resulting in elevated levels of TGF-.beta.1 relative to a normal mouse of the same strain.

5. The transgenic mouse of claim 1, wherein said hAPP is APP770.

6. The transgenic mouse of claim 1, wherein said hAPP is APP751.

7. The transgenic mouse of claim 1, wherein said hAPP is APP695.

8. The transgenic mouse of claim 1, wherein said hAPP is a mutant hAPP.

9. The transgenic mouse of claim 8, wherein said mutant hAPP comprises a familialAD mutation.

10. The transgenic mouse of claim 8, wherein said hAPP mutant is APP695 comprising a valine to isoleucine substitution at amino acid 642.

11. The transgenic mouse of claim 8, wherein said hAPP mutant is APP695 comprising a valine to phenylalanine substitution at amino acid 642.

12. The transgenic mouse of claim 8, wherein said hAPP mutant is APP695 comprising a valine to glycine substitution at amino acid 642.

13. A method of screening for biologically active agents that modulate a phenomenon associated with Alzheimer's disease (AD), the method comprising:

combining a candidate agent with a transgenic mouse comprising a transgenic nucleotide sequence encoding bioactive transforming growth factor-.beta.1 (TGF-.beta.1) operably linked to a promoter and stably integrated into the genome of said mouse, wherein said nucleotide sequence is expressed and wherein said expression results in cerebrovascular amyloid deposits associated with AD; and

determining the effect of said agent upon a phenomenon associated with AD.

14. The method of claim 13, wherein the transgenic mouse further comprises a second transgenic nucleotide sequence encoding a human amyloid precursor protein (hAPP) gene product, wherein said second nucleotide sequence is expressed and wherein expression of said second nucleotide sequence results in development, within about three months of age, of cerebrovascular amyloid deposits associated with AD in said mouse.

15. The method of claim 13, wherein the phenomenon associated with Alzheimer's disease is amyloid deposition.

16. The method of claim 13, wherein the phenomenon associated with Alzheimer's disease is production of a proteolytic fragment of hAPP selected from the group consisting of A.beta..sub.1-40 and A.beta..sub.1-42.

17. The method of claim 13, wherein the phenomenon associated with Alzheimer's disease is neuronal cell loss.

18. A method of screening for biologically active agents that modulate a phenomenon associated with cerebral amyloid angiopathy (CAA), the method comprising:

combining a candidate agent with a transgenic mouse comprising a transgenic nucleotide sequence encoding bioactive transforming growth factor-.beta.1 (TGF-.beta.1) operably linked to a promoter and stably integrated into the genome of said mouse, wherein said nucleotide sequence is expressed and wherein said expression results in cerebrovascular amyloid deposits associated with CAA; and

determining the effect of said agent upon a phenomenon associated with CAA.

19. The method of claim 18, wherein the transgenic mouse further comprises a second transgenic nucleotide sequence encoding a human amyloid precursor protein (hAPP) gene product stably integrated into the genome of said mouse, wherein said second nucleotide sequence is expressed and wherein said animal develops, within about three months of age, cerebrovascular amyloid deposits associated with CAA.

20. The method of claim 16, wherein the phenomenon associated with cerebral amyloid angiopathy is cerebrovascular amyloid deposition.

21. The method of claim 16, wherein the phenomenon associated with cerebral amyloid angiopathy is cerebral hemorrrhage.

22. A method of screening for biologically active agents that modulate a pathology associated with Alzheimer's disease (AD), the method comprising:

combining a candidate agent with a transgenic mouse comprising stably integrated into the genome of said mouse a first transgenic nucleotide sequence encoding bioactive transforming growth factor-.beta.1 (TGF-.beta.1) operably linked to a promoter and a second transgenic nucleotide sequence encoding a human amyloid

precursor protein (hAPP) operably linked to a promoter, wherein said first and said second transgenic nucleotide sequences are expressed, and wherein, as a result of said expression, said transgenic animal develops, within about three months of age, cerebrovascular amyloid deposits associated with AD; and

determining the effect of said agent upon a pathology associated with AD.

23. A method of screening for biologically active agents that modulate a pathology associated with cerebral amyloid angiopathy (CAA), the method comprising:

combining a candidate agent with a transgenic mouse comprising stably integrated into the genome of said mouse a first transgenic nucleotide sequence encoding bioactive transforming growth factor-.beta.1 (TGF-.beta.1) operably linked to a promoter and a second transgenic nucleotide sequence encoding a human amyloid precursor protein (hAPP) operably linked to a promoter, wherein said first and said second transgenic nucleotide sequences are expressed, and wherein, as a result of said expression, said transgenic mouse develops, within about three months of age, cerebrovascular amyloid deposits associated with CAA; and

determining the effect of said agent upon a pathology associated with CAA.

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L3: Entry 11 of 18 File: USPT Mar 2, 1999

US-PAT-NO: 5877399
DOCUMENT-IDENTIFIER: US 5877399 A

TITLE: Transgenic mice expressing APP-Swedish mutation develop progressive neurologic disease

DATE-ISSUED: March 2, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hsiao; Karen	North Oaks	MN		
Borchelt; David R.	Baltimore	MD		
Sisodia; Sangram S.	Baltimore	MD		

US-CL-CURRENT: 800/3; 424/9.2, 800/12, 800/9

CLAIMS:

What is claimed is:

1. A transgenic mouse whose genome comprises:

an amyloid precursor protein transgene comprising regulatory sequences from a prion gene promoter operatively linked to a coding sequence selected from the group consisting of APP695.WT, APP695.SWE and APP695.TRI, wherein neurological expression of said transgene produces impaired performance of said mouse in memory and learning tests and induces abnormal neuropathology in a cortico-limbic region of said mouse's brain, wherein said impaired performance and said abnormal neuropathology are in comparison with control mice.

2. The transgenic mouse according to claim 1, wherein said transgene expression produces levels of amyloid percursor protein at least about two-fold that of endogenous amyloid precursor protein in a control mouse.

3. The transgenic mouse according to claim 1, wherein said abnormal neuropathology is thioflavin S positive amyloid plaques.

4. The transgenic mouse according to claim 1, wherein a nontransgenic ancestor of said mouse is from a strain having greater longevity as compared with other strains of mice.

5. Progeny of said transgenic mice according to claim 1, wherein the genome of the progeny comprises an amyloid precursor protein transgene comprising regulatory sequences from a prion gene promoter operatively linked to a coding sequence selected from the group consisting of APP695.WT, APP695.SWE and APP695.TRI, wherein neurological expression of said transgene produces impaired performance of said progeny in memory and learning tests and induces abnormal neuropathology in a cortico-limbic region of said progeny's brain, wherein said impaired performance and said abnormal neuropathology are in comparison with control mice.

6. A method for screening for an agent which ameliorates symptoms of Alzheimer's disease, said method comprising:

comparing performance on memory and learning tests of a first transgenic mouse contacted with said agent with that of a second transgenic mouse not contacted with said agent, wherein the genome of said first and said second transgenic mice comprise an amyloid precursor protein transgene comprising regulatory sequences from a prion gene promoter operatively linked to a coding sequence selected from the group consisting of APP695.WT, APP695.SWE and APP695.TRI, wherein neurological expression of said transgene produces impaired performance on said memory learning tests and abnormal neuropathology in a cortico-limbic region of said mouse's brain, wherein said impaired performance and said abnormal neuropathology are in comparison with control mice, whereby an agent which ameliorates said symptoms is identified by superior performance of said first transgenic mouse in comparison with said second transgenic mouse on said memory and learning tests.

7. A method for screening for an agent useful for treating Alzheimer's disease, said method comprising:

comparing performance on memory and learning tests of a first transgenic mouse contacted with said agent with that of a second transgenic mouse not contacted with said agent, wherein the genome of said first and said second transgenic mice comprise an amyloid precursor protein transgene comprising regulatory sequences from a prion gene promoter operatively linked to a coding sequence selected from the group consisting of APP695.WT, APP695.SWE and APP695.TRI, wherein neurological expression of said transgene produces impaired performance on said memory and learning tests and abnormal neuropathology in a cortico-limbic region of said mouse's brain, wherein said impaired performance and said abnormal neuropathology are compared with control mice; and

comparing neuropathology in a cortico-limbic region of the brain of said first and said second transgenic mice when said first transgenic mouse exhibits superior performance on said memory and learning tests compared with said second transgenic mouse, whereby an agent which is useful for treating Alzheimer's disease is identified by a decrease in neuropathologic findings in said first transgenic mouse in comparison with said second transgenic mouse.

8. The method according to claim 7, wherein said decreased neuropathologic findings are one or more findings selected from the group consisting of:

a reduction in number of thioflavin S-positive A.beta. deposits;

a reduction in amount of thioflavin S-positive A.beta. deposits;

a reduction of hypertrophic gliosis in cortico-limbic structures of said brain;

a reduction of diminution of 2-deoxyglucose uptake in cortico-limbic structures of said brain; and

a reduction of diminution of 2-deoxyglucose utilization in cortico-limbic structures of said brain.

9. A method for screening for an agent useful for treating Alzheimer's disease, said method comprising:

comparing performance on memory and learning tests of a first transgenic mouse contacted with said agent with that of a second transgenic mouse not contacted with said agent, wherein the genome of said first and said second transgenic mice comprise an amyloid precursor protein transgene comprising regulatory sequences from a prion gene promoter operatively linked to a coding sequence selected from the group consisting of APP695.WT, APP695.SWE and APP695.TRI, wherein neurological expression of said transgene produces impaired performance on said memory and learning tests and abnormal neuropathology in a cortico-limbic region of said mousers brain, wherein said impaired performance and abnormal neuropathology are in comparison with control mice; and

comparing age of death of said first and said second transgenic mice when said first

transgenic mouse exhibits superior performance on said memory and learning tests in comparison with said second transgenic mouse, whereby an agent which is useful for treating Alzheimer's disease is identified by a greater age at death of said first transgenic mouse in comparison with said second transgenic mouse.

10. A method for screening for an agent which ameliorates symptoms of Alzheimer's disease, said method comprising:

comparing exploratory behavior or locomotor behavior of a first transgenic mouse contacted with said agent with that of a second transgenic mouse not contacted with said agent, wherein the genome of said first and said second transgenic mice comprise an amyloid precursor protein transgene comprising regulatory sequences from a prion gene promoter operatively linked to a coding sequence selected from the group consisting of APP695.WT, APP695.SWE and APP695.TRI, wherein neurological expression of said transgene produces impaired performance on said exploratory and locomotor behavior and abnormal neuropathology in a cortico-limbic region of said mouse's brain, wherein said impaired performance and abnormal neuropathology are compared with control mice, whereby an agent which ameliorates said symptoms is identified by less impaired exploratory or locomotor behavior in said first transgenic mouse in comparison with said second transgenic mouse.

11. The method according to claim 10, wherein said exploratory or locomotor behavior is assessed using a corner index test.

12. The method according to claim 10, wherein a nontransgenic ancestor of said mouse is from a strain selected from the group consisting of FVB, Swiss Webster, and C57B6.

13. A transgenic mouse with progressive neurological disease whose genome comprises:

an amyloid precursor protein transgene comprising regulatory sequences from a prion gene promoter operatively linked to an amyloid precursor protein coding sequence having the Swedish mutation, wherein neurological expression of said transgene produces an age-dependent decline in performance of said mouse in memory and learning tests and produces amyloid plaques that are detectable by Congo red staining in the brain of said mouse, wherein said age-dependent decline in performance and said amyloid plaques are in comparison with control mice.

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L3: Entry 12 of 18 File: USPT Dec 15, 1998

US-PAT-NO: 5850003
DOCUMENT-IDENTIFIER: US 5850003 A

TITLE: Transgenic rodents harboring APP allele having swedish mutation

DATE-ISSUED: December 15, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
McLonlogue; Lisa C.	San Francisco	CA		
Zhao; Jun	San Diego	CA		

US-CL-CURRENT: 800/9; 800/12, 800/14, 800/18

CLAIMS:

We claim:

1. A transgenic rodent comprising a diploid genome comprising a transgene comprising in operable linkage a promoter, a DNA segment encoding a heterologous APP polypeptide and a polyadenylation site, wherein the APP polypeptide has the Swedish mutation whereby the amino acid residues at positions corresponding to positions 595 and 596 in human APP695 are asparagine and leucine, respectively, wherein the transgene is expressed to produce a human APP polypeptide having the Swedish mutation, and wherein said polypeptide is processed to ATF-betaAPP in a sufficient amount to be detectable in a brain homogenate of said transgenic rodent.

2. A transgenic rodent of claim 1, wherein the rodent is a mouse.

3. A transgenic rodent of claim 1, wherein the transgene is nonhomologously integrated.

4. A transgenic rodent of claim 1, wherein the promoter is a neural-specific enolase promoter.

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L3: Entry 13 of 18 File: USPT Mar 18, 1997

US-PAT-NO: 5612486
DOCUMENT-IDENTIFIER: US 5612486 A

TITLE: Transgenic animals harboring APP allele having swedish mutation

DATE-ISSUED: March 18, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
McConlogue; Lisa C.	San Francisco	CA		
Zhao; Jun	San Diego	CA		

US-CL-CURRENT: 800/12; 536/23.1, 536/23.5, 800/18

CLAIMS:

We claim:

1. A transgenic rodent comprising a diploid genome comprising a transgene encoding a heterologous APP polypeptide having the Swedish mutation wherein the amino acid residues at positions corresponding to positions 595 and 596 in human APP695 are asparagine and leucine, respectively, wherein the transgene is expressed to produce a human APP polypeptide having the Swedish mutation, and wherein said polypeptide is processed to ATF-betaAPP in a sufficient amount to be detectable in a brain homogenate of said transgenic rodent.

2. A transgenic rodent of claim 1, wherein the animal is murine.

3. A transgenic rodent of claim 2, wherein the transgene is nonhomologously integrated.

4. A transgenic rodent of claim 1, wherein the heterologous APP polypeptide having the Swedish mutation is expressed under the transcriptional control of a neural-specific enolase promoter.

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☐ 11. Document ID: US 5877399 A

L3: Entry 11 of 18 File: USPT Mar 2, 1999

US-PAT-NO: 5877399
DOCUMENT-IDENTIFIER: US 5877399 A

TITLE: Transgenic mice expressing APP-Swedish mutation develop progressive neurologic disease

DATE-ISSUED: March 2, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hsiao; Karen	North Oaks	MN		
Borchelt; David R.	Baltimore	MD		
Sisodia; Sangram S.	Baltimore	MD		

US-CL-CURRENT: 800/3; 424/9.2, 800/12, 800/9

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 12. Document ID: US 5850003 A

L3: Entry 12 of 18 File: USPT Dec 15, 1998

US-PAT-NO: 5850003
DOCUMENT-IDENTIFIER: US 5850003 A

TITLE: Transgenic rodents harboring APP allele having swedish mutation

DATE-ISSUED: December 15, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
McLonlogue; Lisa C.	San Francisco	CA		
Zhao; Jun	San Diego	CA		

US-CL-CURRENT: 800/9; 800/12, 800/14, 800/18

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 13. Document ID: US 5612486 A

L3: Entry 13 of 18 File: USPT Mar 18, 1997

US-PAT-NO: 5612486
DOCUMENT-IDENTIFIER: US 5612486 A

TITLE: Transgenic animals harboring APP allele having swedish mutation

DATE-ISSUED: March 18, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
McConlogue; Lisa C.	San Francisco	CA		
Zhao; Jun	San Diego	CA		

US-CL-CURRENT: 800/12; 536/23.1, 536/23.5, 800/18

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

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☐ 14. Document ID: US 5604102 A

L3: Entry 14 of 18 File: USPT Feb 18, 1997

US-PAT-NO: 5604102
DOCUMENT-IDENTIFIER: US 5604102 A

TITLE: Methods of screening for .beta.-amyloid peptide production inhibitors

DATE-ISSUED: February 18, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
McConlogue; Lisa C.	San Francisco	CA		
Schenk; Dale B.	Pacifica	CA		
Seubert; Peter A.	South San Francisco	CA		
Sinha; Sukanto	San Francisco	CA		
Zhao; Jun	La Jolla	CA		

US-CL-CURRENT: 435/7.1; 424/9.2, 435/7.21, 530/350

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☐ 15. Document ID: US 5877399 A

L3: Entry 15 of 18 File: EPAB Mar 2, 1999

PUB-NO: US005877399A
DOCUMENT-IDENTIFIER: US 5877399 A
TITLE: Transgenic mice expressing APP-Swedish mutation develop progressive neurologic disease

PUBN-DATE: March 2, 1999

INVENTOR-INFORMATION:

NAME	COUNTRY
HSIAO, KAREN	US
BORCHELT, DAVID R	US
SISODIA, SANGRAM S	US

INT-CL (IPC): C12 N 5/00; C12 N 15/00; A61 K 49/00

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
Draw Desc | Image

☐ 16. Document ID: US 5850003 A

L3: Entry 16 of 18 File: EPAB Dec 15, 1998

PUB-NO: US005850003A
DOCUMENT-IDENTIFIER: US 5850003 A
TITLE: Transgenic rodents harboring APP allele having swedish mutation

PUBN-DATE: December 15, 1998

INVENTOR-INFORMATION:

NAME	COUNTRY
MCLONLOGUE, LISA C	US
ZHAO, JUN	US

INT-CL (IPC): C12 N 5/00; C12 N 15/00

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
Draw Desc | Image

☐ 17. Document ID: WO 9803644 A1

L3: Entry 17 of 18 File: EPAB Jan 29, 1998

PUB-NO: WO009803644A1
DOCUMENT-IDENTIFIER: WO 9803644 A1
TITLE: TRANSGENIC ANIMAL MODEL FOR ALZHEIMER DISEASE

PUBN-DATE: January 29, 1998

INVENTOR-INFORMATION:

NAME	COUNTRY
SOMMER, BERND	DE
STAUFENBIEL, MATTHIAS	DE

INT-CL (IPC): C12 N 15/00; A01 K 67/027; C07 K 14/47; C12 N 15/12; A61 K 49/00
EUR-CL (EPC): A01K067/027; C07K014/47

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☐ 18. Document ID: US 5612486 A

L3: Entry 18 of 18 File: EPAB Mar 18, 1997

PUB-NO: US005612486A
DOCUMENT-IDENTIFIER: US 5612486 A
TITLE: Transgenic animals harboring APP allele having swedish mutation

PUBN-DATE: March 18, 1997

INVENTOR-INFORMATION:

NAME	COUNTRY
MCCONLOGUE, LISA C	US
ZHAO, JUN	US

INT-CL (IPC): C12 N 15/00; C07 H 21/04
EUR-CL (EPC): C07K014/47; C07K016/18, A01K067/027

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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Terms	Documents
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☐ 1. Document ID: US 20020022002 A1

L3: Entry 1 of 18 File: PGPB Feb 21, 2002

PGPUB-DOCUMENT-NUMBER: 20020022002
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020022002 A1

TITLE: Methods for labeling beta-amyloid plaques and neurofibrillary tangles

PUBLICATION-DATE: February 21, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Barrio, Jorge R.	Agoura Hills	CA	US	
Petric, Andrej	Ljubljana	CA	SI	
Satyamurthy, Nagichettiar	Los Angeles	CA	US	
Small, Gary W.	Los Angeles	CA	US	
Cole, Gregory M.	Santa Monica	CA	US	
Huang, Sung-Cheng	Sherman Oaks		US	

US-CL-CURRENT: 424/1.37; 435/40.5

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

☐ 2. Document ID: US 20020019992 A1

L3: Entry 2 of 18 File: PGPB Feb 14, 2002

PGPUB-DOCUMENT-NUMBER: 20020019992
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020019992 A1

TITLE: TRANSGENIC NON-HUMAN MAMMALS WITH PROGRESSIVE NEUROLOGIC DISEASE

PUBLICATION-DATE: February 14, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
HSIAO, KAREN	NORTH OAKS	MN	US	
BORCHELT, DAVID R.	BALTIMORE	MD	US	
SISODIA, SANGRAM	BALTIMORE	MD	US	

US-CL-CURRENT: 800/3; 800/13, 800/14, 800/18

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☐ 3. Document ID: US 20020010947 A1

L3: Entry 3 of 18 File: PGPB Jan 24, 2002

PGPUB-DOCUMENT-NUMBER: 20020010947
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020010947 A1

TITLE: Transgenic mouse model of human neurodegenerative disease

PUBLICATION-DATE: January 24, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Gurney, Mark E.	Reykjavik	WA	IS	
Abraham, Irene	Seattle		US	

US-CL-CURRENT: 800/12; 800/18

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☐ 4. Document ID: US 20010016951 A1

L3: Entry 4 of 18 File: PGPB Aug 23, 2001

PGPUB-DOCUMENT-NUMBER: 20010016951
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20010016951 A1

TITLE: Transgenic animal model for alzheimer disease

PUBLICATION-DATE: August 23, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Sommer, Bernd	Eimeldingen		DE	
Staufenbiel, Matthias	Loerrach-Haagen		DE	

US-CL-CURRENT: 800/3; 800/12, 800/18

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☐ 5. Document ID: US 6313268 B1

L3: Entry 5 of 18 File: USPT Nov 6, 2001

US-PAT-NO: 6313268
DOCUMENT-IDENTIFIER: US 6313268 B1

TITLE: Secretases related to Alzheimer's dementia

DATE-ISSUED: November 6, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hook; Vivian Y. H.	La Jolla	CA	92037	

US-CL-CURRENT: 530/350; 424/563, 424/570, 424/94.1, 424/94.2, 424/94.6, 424/94.63, 424/94.66, 435/183, 435/212, 435/226 , 530/412, 530/422, 530/427

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☐ 6. Document ID: US 6274119 B1

L3: Entry 6 of 18 File: USPT Aug 14, 2001

US-PAT-NO: 6274119
DOCUMENT-IDENTIFIER: US 6274119 B1

TITLE: Methods for labeling .beta.-amyloid plaques and neurofibrillary tangles

DATE-ISSUED: August 14, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Barrio; Jorge R.	Agoura Hills	CA		
Petric; Andrej	Ljubljana			SIX
Satyamurthy; Nagichettiar	Los Angeles	CA		
Small; Gary W.	Los Angeles	CA		
Cole; Gregory M.	Santa Monica	CA		
Huang; Sung-Cheng	Sherman Oaks	CA		

US-CL-CURRENT: 424/1.81; 424/1.85

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☐ 7. Document ID: US 6245964 B1

L3: Entry 7 of 18 File: USPT Jun 12, 2001

US-PAT-NO: 6245964
DOCUMENT-IDENTIFIER: US 6245964 B1

TITLE: Transgenic rodent comprising APP-Swedish

DATE-ISSUED: June 12, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
McLonlogue; Lisa C.	San Francisco	CA		
Zhao; Jun	La Jolla	CA		
Sinha; Sukanto	San Francisco	CA		

US-CL-CURRENT: 800/12; 800/14, 800/18, 800/22, 800/3

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☐ 8. Document ID: US 6245884 B1

L3: Entry 8 of 18 File: USPT Jun 12, 2001

US-PAT-NO: 6245884
DOCUMENT-IDENTIFIER: US 6245884 B1

TITLE: Secretases related to alzheimer's dementia

DATE-ISSUED: June 12, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hook; Vivian Y. H.	La Jolla	CA	92037	

US-CL-CURRENT: 530/300; 424/9.2, 435/23, 435/29, 435/326, 435/331, 435/332, 435/69.2, 435/7.1, 530/331, 530/350, 562/545 , 562/577

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☐ 9. Document ID: US 6175057 B1

L3: Entry 9 of 18 File: USPT Jan 16, 2001

US-PAT-NO: 6175057
DOCUMENT-IDENTIFIER: US 6175057 B1

TITLE: Transgenic mouse model of alzheimer's disease and cerebral amyloid angiopathy

DATE-ISSUED: January 16, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Mucke; Lennart	Foster City	CA		
Wyss-Coray; Tony	Berkeley	CA		
Masliah; Eliezer	Chula Vista	CA		

US-CL-CURRENT: 800/12; 424/9.2, 800/18, 800/3

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC
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☐ 10. Document ID: US 6114133 A

L3: Entry 10 of 18 File: USPT Sep 5, 2000

US-PAT-NO: 6114133
DOCUMENT-IDENTIFIER: US 6114133 A

TITLE: Methods for aiding in the diagnosis of Alzheimer's disease by measuring amyloid-.beta. peptide (x-.gtoreq.41)

DATE-ISSUED: September 5, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Seubert; Peter A.	South San Francisco	CA		
Vigo-Pelfrey; Carmen	Mountain View	CA		
Schenk; Dale B.	Pacifica	CA		
Barbour; Robin	Newark	CA		

US-CL-CURRENT: 435/7.94; 435/7.1, 435/7.92, 436/518, 436/811

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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